Lower Cuticle Temperature Over Time Leads to Faster Fingernail Growth; Implications for Role of Temperature Sensing Molecules in Body in Up and Down-Regulation of Stem Cells

13 March 2024
Simon Edwards
Research Acceleration Initiative

Introduction

Cold-blooded creatures including the salamander are known for their remarkable ability to re-grow severed limbs and are also known for their dependence upon warm conditions in order for biological functions to be enabled. Unable to generate substantial body heat, cold-blooded creatures hibernate at temperatures substantially below room temperature.

Warm-blooded animals are capable of generating greater amounts of body heat and are therefore able to function in a wider variety of climates including cold climates. When considering warm-blooded creatures, warmth is often associated with pro-growth conditions and coldness is often associated with conditions which stunt growth. This paradigm is nothing but a misconception and has regrettably, in this author's view, frustrated efforts at discovering a means of duplicating the healing ability of the salamander.

Abstract

The genetic and epigenetic switches that determine a person's ultimate height, for example, are generally thought to be governed by the availability of nutrients. It is taught in schools that multiple, successive generations having access to plentiful food has resulted in children growing to be taller than their parents.

While a paucity of access to nutrients would certainly have the effect of stunting growth and activating epigenetic switches which trigger slowed metabolism, a different factor is likely driving the generational tendency toward taller individuals. That factor is the exposure to cold weather conditions whilst having sufficient available nutrition to support growth. I propose that the single greatest factor in determining whether a child (provided that at least adequate nutrition is available) grows to their maximal height potential is the proportion of time they spend in an uncomfortably cold environment. A difference of even a few degrees on a household thermostat in the winter could dramatically increase the ultimate height of a child reared in that household provided that comparatively cool conditions prevail over a period of years.

The best evidence to support this contention would be a simple study of fingernail growth under cold conditions. If one were to study the rate of fingernail growth on one hand versus another when one hand is kept artificially cold over a period of several hours per day, at the end of a week, the researchers

would find that the colder fingernails grew to a longer length in the same length of time as the "warm" nails.

If this is experimentally verified (it would cost virtually nothing to perform such an experiment,) it would be sensible to investigate the possibility that it is, in fact, a biochemical perception of excessive heat which inhibits growth. The root cells in the cuticles from which nails grow are examples of stem cells which remain active throughout life and crucially, they are known examples of cells which remain active even days after death. Despite decreased access to nutrients and no flow of blood, nails and hair continue to grow (in fact, at an accelerated rate) in deceased humans for a period of days after the cessation of life. This disproportional growth associated with the nails and hair of the dead is further support for the contention that the presence of an mRNA signal emanating from a 'thermostat molecule' (first proposed by this author in 2021) actively suppresses growth and that in its absence, cellular generation in all stem cells would be intensely accelerated. It is not the temperature, itself which determines the growth rate but rather the presence or absence of specific mRNA signals. This contention could be confirmed by comparing the growth rate of the fingernails of corpses stored at differing temperatures. In my hypothesis, activity rates should be roughly the same for the follicles of both cold and warm corpses given that the absence of blood flow renders as zero the influence of the mRNA down-regulation of that activity.

One possible explanation for this dynamic is the presence of the previously speculated temperature-sensing molecules in the bloodstream (ibid. paper regarding dynamics of carcinogenesis related to cell phone use/microwaves.) When blood is not circulating, these temperature-sensing molecules would not be locally bio-available and would therefore no longer serve, under that circumstance, to down-regulate cellular production by stem cells (provided that they exist and are down-regulators of cellular production) regardless of the actual temperature.

Given that growth is a process which contributes to higher body temperatures, the need for biological growth must be balanced with both nutrient availability as well as a need to maintain thermal homeostasis. If it were not for the need to maintain body temperature below a certain level (to prevent damage to the brain,) creatures might gain evolutionary advantage by gorging themselves when food supplies are abundant and experiencing phenomenal levels of growth at the expense of extreme increases in body temperature. Such dramatic increases in body temperature are disruptive to other biological functions such as healthy brain function. This need to balance thermal homeostasis and the need to have "growth spurts" which take advantage of transiently available abundant food supplies is likely the evolutionary reason why growth rates are accelerated by decreased temperatures in humans, primates and perhaps mammals, as well. When experienced temperatures are cold over a period of at least several hours per day, the body senses that a growth spurt is biologically safe. While the conventional wisdom holds that humans grow when nutrients are available, what is actually occurring is more nuanced. *Animals store metabolic energy and*

utilize invest that energy in growth when body temperature is gauged to be comparatively low.

This is good opportunity to point out (something which has likely been pointed out previously) that infections lead to fever as a result of the heat generated by the cellular production process (both bacterial cells and white blood cell production) in contrast to the institutionally-accepted and promoted misconception that fevers are some sort of evolved defense mechanism against infection which assumes that an increase in body temperature of a mere few degrees Fahrenheit would be sufficient to "cook" bacteria. Such a notion is easy to identify as fallacious given that bacterial growth is not inhibited in any way until temperatures exceed 120 Fahrenheit. The febrile state associated with infection is a symptom of infection and the heat generation resulting from the necessary cell production associated with the immune response. Keeping a patient cold (although highly uncomfortable for a sick person) would likely do more to promote a given patient's recovery (irrespective of antibiotic availability) given that it would enable the more efficient production of white blood cells and would slow the replication rate of the bacteria associated with infection.

For humans, something as dramatic as the re-growth of a severed limb would require the addition of stem cells to the affected area where no stem cells would ordinarily be found. In the case of generic injuries, however, fibroblast cells are required in order to support the regrowth of skin and soft tissue. The deformation or paucity of these cells can lead to delayed and improper healing. It is well-understood that children have a superior ability to heal "properly" and that the older someone is, the longer it takes for a wound to heal, all other things being equal. It may be possible that the poor circulation and decreased body temperature associated with advancing age could be partially attributable to increased expression of mRNA from the 'thermostat molecule' and that this overexpression is what frustrates fibroblastic support of wound healing in the elderly.

A complication faced in experimental stem cell therapies aimed at tissue regeneration is the difficulty experienced by researchers in reliably "turning off" the cellular production phase of the stem cells after they have performed the desired task. As tissue regeneration after an injury is not a one-size-fits-all proposition, standardized patterns of activation and deactivation of stem cells cannot be used to restore the original characteristics of damaged tissues and organs. To accomplish this, one would have to hyper-accurately pre-program the therapy to account for both the damaged state of the tissue and the desired restored state. This could well-prove to make stem cell-based tissue regeneration (in cases of complex injury to a tissue or organ) to be an impractically complex proposition.

The challenged faced in the field is no longer one of producing sufficient numbers of stem cells but rather in deactivating them after they have performed their task so as to make the therapy safe. We can infer from the aforementioned deductions that stem cells may be controllably deactivated through the

introduction of synthetic forms of the RNA associated with the heretofore unidentified temperature-sensing molecules of the bloodstream. One this molecule is identified and its mRNA signature is evaluated, one would be able to use the signal associated with "high" temperature in order to deactivate stem cells. Small doses of this synthesized mRNA could be used to temporarily halt cell growth with large doses being useful for tricking the stem cells into morphing into permanent cells of the surrounding type which cannot produce additional cells. The phenomena of both hair loss and graying hair associated with a geriatric state could be explained by the overexpression of mRNA by the heretofore unidentified 'thermostat molecule' in old age. This further serves to support the author's overall contentions.

Conclusion

This insight should provide yet another strong incentive for the research community to begin to undertake research aimed at identifying the temperature-sensing bio-molecule likely to exist in the human bloodstream which is relevant both for the regulation of growth both in terms of gestation and in terms of healing as well as in triggering undesired illnesses such as cancers brought about by exposure to microwave energy. Remarkably, there has been no research, as of yet, into the possible existence of such a molecule. Whichever molecule is responsible is likely to have a lipid membrane given that lipids convert microwave energy more efficiently into heat than water molecules can, given our previous hypothesis on the topic of microwave-induced carcinogenesis (ibid.) This heretofore unidentified molecule could be a molecule already known to biologists which performs some other function and thus performs a 'thermostat function' secondarily to some other known function, resulting in a collective failure, heretofore, to identify it.